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Bayesian synthesis of a pathogen growth model: *Listeria monocytogenes* under competition☆

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Abstract

The Bayesian synthesis method is applied to data from two studies of *Listeria monocytogenes* grown in broth monocultures to draw inferences about the joint distribution of two Baranyi growth model parameters–lag time and maximum specific growth rate. The resultant joint distribution is then combined with prior distributions for the initial and maximum pathogen density parameters under competitive growth conditions. Finally, the pathogen growth model is updated using the Sampling/Importance Resampling (SIR) algorithm with data on *L. monocytogenes* growth in competition with natural microflora in fish. Although the latter data provide no information on the stationary phase to directly estimate the maximum pathogen density parameter, combining them with relevant prior information provides a means to characterize *L. monocytogenes* growth in a food with mixed microbial populations. Based on a specified tolerance for *L. monocytogenes* growth, the updated model provides a storage time limit for fish held at 5 °C, pH 6.8, 43% CO_2, 57% N_2.

Keywords: Predictive microbiology; *Listeria monocytogenes*; Competitive growth; Bayesian analysis

1. Introduction

A variety of approaches have been proposed to evaluate and compare the performance of predictive microbiology models (Baranyi et al., 1999; Delignette-Muller et al., 1995; Duh and Schaffer, 1993; McClure et al., 1993; Ross, 1996; Wijtzes et al., 1993). Each of these methods presumes, implicitly or otherwise, that an independent "gold standard" or benchmark data set is available. In most risk assessment applications, however, ideal data with which to compare model predictions are unavailable. This problem is amplified when considering the variability and uncertainty associated with microbial community dynamics in food (Powell et al., 2004). As an alternative or complement to the model validation approach, Bayesian statistical methods such as Bayesian synthesis (Raftery et al., 1995) and Sampling/Importance Resampling (Rubin, 1988) offer means of combining information from a variety of sources (laboratory experiments, field measurements, and/or expert judgment) to develop and evaluate predictive microbiology models for use in microbial pathogen risk assessment.

Raftery et al. (1995) first proposed the Bayesian synthesis method to characterize uncertainty in mechanistic process models (e.g., of population dynamics). A key feature of Bayesian synthesis is that it assumes that prior information on both the inputs and outputs is available from independent information, as are the empirical data used to update the model. In contrast, conventional Bayesian methods only permit incorporation of information on model outputs as empirical data to update the model. A principal advantage of Bayesian synthesis, therefore, is that it permits incorporation of available prior information on outputs as well as inputs of the model.

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The basic approach in Bayesian synthesis is to generate a joint prior distribution on all model inputs and outputs. A joint posterior distribution on the inputs and outputs is then generated by importance sampling (i.e., weighted sampling). The joint posterior distribution can be marginalized to obtain posterior distributions on the inputs and outputs, as well as the estimated correlation structure among the model parameters. Both Bayesian synthesis and Markov Chain Monte Carlo (MCMC) methods (e.g., the Metropolis–Hastings algorithm and Gibbs sampling) are Bayesian Monte Carlo procedures, which update uncertainty with backward and forward propagation of the model (Brand and Small, 1995). This feature permits the analyst to characterize available, imperfect data on model inputs as prior distributions to be updated based on empirical evidence on the model output. In contrast to the non-iterative importance sampling of Bayesian synthesis, MCMC methods are based on an iterative updating scheme that is repeated until the sequence of parameter vectors converges. Both importance sampling and MCMC methods may be computationally intensive; however, MCMC methods also may fail to converge to the stationary distribution, e.g., converging to local rather than global maxima of the likelihood function due to a multimodal probability surface (Robert and Casella, 1999).

This paper uses the Bayesian synthesis method to combine informed prior distributions for pathogen growth model inputs with data from two studies on *Listeria monocytogenes* growth in broth monoculture. The result of this synthesis is then combined with other information to characterize *L. monocytogenes* growth in a mixed microbial community in a food stored under modified atmosphere (i.e., reduced oxygen) conditions and refrigeration temperatures. *L. monocytogenes* can be isolated from a wide variety of environmental sources and animal reservoirs, including asymptomatic humans, can grow at refrigeration temperatures, and tolerates salt, freezing, drying, and a wide range of pH conditions. Because *L. monocytogenes* is susceptible to cooking and pasteurization, cross-contamination of ready-to-eat food is the normal route of transmission in processed foods. Modified atmosphere packaging combined with refrigeration is a popular preservation technique for minimally processed food because it inhibits the growth of aerobic spoilage bacteria (e.g., *Pseudomonas* spp.) and oxidative processes. *L. monocytogenes* is still able to grow, however, at low temperatures in 100% carbon dioxide (CO_2) (Pin et al., 2001). In the United States, the population-based incidence of listeriosis is low relative to other foodborne pathogens (e.g., Norwalk-like viruses, *Salmonella*, and *Campylobacter*). Among illnesses due to foodborne pathogens, however, listeriosis has one of the highest case-fatality rates (approximately 20%), particularly in susceptible subpopulations—the elderly and perinatal age groups and immunocompromised individuals. Listeriosis can result in septicemia, meningitis, and perinatal miscarriage, stillbirth, or meningitis. Listerial gastroenteritis, a milder form of *L. monocytogenes* infection resulting in flu-like symptoms, has recently been recognized (note that the reported case-fatality rate is based on the more severe listeriosis case definition). In the United States, deli meats, frankfurters (not reheated), and pâté and meat spreads are estimated to pose the highest risk of listeriosis on a per serving basis, while the highest burden of listeriosis on a per annum basis has been attributed to deli meats (Health and Consumer Protection Directorate, 1999; Centers for Disease Control and Prevention, 2003; Center for Food Safety and Applied Nutrition, 2003; Food Safety and Inspection Service, 2003).

The plan for this paper is as follows: Sec. 2) the Baranyi pathogen growth model is specified; Sec. 3) informed priors on the Baranyi model inputs provide an implied prior on *L. monocytogenes* growth in monoculture at 5 °C, pH 7; Sec. 4) data reported by Buchanan et al. (1989) provide an independent stated prior on *L. monocytogenes* growth at 5 °C, pH 7 in broth monoculture; Sec. 5) data reported by Pin et al. (2001) on *L. monocytogenes* growth at 5 °C, pH 7 in broth monoculture under elevated CO_2 levels are introduced; Sec. 6) a Bayesian synthesis algorithm is used to update the Baranyi model inputs and outputs for *L. monocytogenes* growth in broth monoculture at 5 °C, pH 7 under elevated CO_2 levels; Sec. 7) data reported by the Universidad Complutense de Madrid, Spain on *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2 are introduced; Sec. 8) the updated joint distribution of two Baranyi model parameters–lag time and maximum specific growth rate—obtained from the Bayesian synthesis procedure is combined with prior distributions for initial and maximum population density for pathogen growth in mixed culture, and the observed data are used to update the Baranyi model for *L. monocytogenes* growth in food with mixed microbial populations using the Sampling/Importance Resampling (SIR) algorithm; Sec. 9) sensitivity analysis; Sec. 10) results; and Sec. 11) discussion.

2. Pathogen growth model

Baranyi and Roberts (1994) originally introduced the Baranyi pathogen growth model. It has been parameterized as follows (Wilson, 1999):

$$y_t = y_0 + \frac{y_1}{\ln(10)} - \frac{y_2}{\ln(10)}$$

where

$$y_t = \log_{10}\text{cfu/g} \text{ at time } t(\text{h})$$

$$y_0 = \log_{10}\text{cfu/g at } t = 0$$

$$y_1 = \mu t + \ln\left[e^{-\mu t} - e^{-\mu (t+t_{\text{lag}})} + e^{-\mu t_{\text{lag}}}\right]$$

$$y_2 = \ln\left[1 + 10^{(y_0 - y_{\max})}\left(e^{\mu (t-t_{\text{lag}})} - e^{-\mu t_{\text{lag}}}\right)\right]$$

$$y_{\max} = \text{ maximum population density } (\log_{10}\text{cfu/g})$$

$$t_{\text{lag}} = \text{ lag time(h)}$$

$$\mu = \text{ maximum specific growth rate}(\text{h}^{-1}) \quad (1)$$

In this form, the parameters of the Baranyi growth model have intuitive biological interpretations, which facilitates specification of their prior distributions. In this paper, we consider the Baranyi growth model without residual variance (i.e., the model contains no error term). Therefore, the output distribution characterizes uncertainty about the mean growth response (due to parameter uncertainty), but does not account for natural or unexplained variability about the estimated growth curve.

3. Prior distributions for model inputs of pathogen growth in monoculture

Broad informed prior distributions on the Baranyi model inputs are specified to capture the entire range of feasible parameter values under monoculture growth conditions. Here, we consider *L. monocytogenes* growth in broth monoculture at 5°C, pH 7.

3.1. Maximum specific growth rate (μ)

According to an international panel of microbiological food safety experts, 29 h is a typical generation time (GT, the time required for population-doubling) for *L. monocytogenes* at 5 °C, pH 7 (Food and Agriculture Organization of the United Nations, 1999). A GT value of 29 h corresponds to a maximum specific growth rate of $\mu = 0.0239$ h^{-1}. The GT point estimate referenced by Food and Agriculture Organization of the United Nations (1999) was based on a model developed by Tienungoon at the University of Tasmania, Australia (Ross et al., 2000). A search of the Combined Database (ComBase) of Predictive Microbiology Information (www.combase.cc; Baranyi and Tamplin, 2004) was conducted to characterize the full range of maximum specific growth rate values for *L. monocytogenes* at 5 °C, pH 7. A query of ComBase for *L. monocytogenes* grown at 4–6 °C with $\mu > 0$ (to filter out lethality treatment datasets) generated 469 values of μ with a mean of 0.0362 h^{-1} (GT = 19.1 h) and a standard deviation (stdev) of 0.0203. A lognormal distribution with these parameter values has a mode of 0.0240 h^{-1} (GT = 28.9 h). Therefore, this distribution was judged consistent with the expert-based estimate of a typical μ value, while capturing the parameter's full range of potential values under the pathogen growth model (i.e., $0 \leq \mu < \infty$).

3.2. Lag time (t_{lag})

Based on a literature review, Ross (1999) observed that the distribution of t_{lag} relative to μ peaks in the range of 4–6 GT equivalents and has an upper 95th percentile in the range of 10–15 GT equivalents. Based on the sample mean of μ obtained above (0.0362 h^{-1}), a lognormal distribution with a mean of 4 GT equivalents and a 95th percentile of 10 GT equivalents has mean value of 76.4939 h and stdev of 61.0093. The lognormal distribution captures the parameter's full range of potential values under the model (i.e., $0 \leq t_{lag} < \infty$).

Table 1
Prior distributions for Baranyi growth model input parameters for *L. monocytogenes* growth at 5 °C, pH 7 in broth monoculture

Input	Distribution	Dist. parameters	95% confidence interval	Units
y_0	Uniform	Min = 2, Max = 4	N/A	$\log_{10}$ cfu/g
y_{max}	Normal	Mean = 9, Stdev = 1	7.04–10.96	$\log_{10}$ cfu/g
t_{lag}	Lognormal	Mean = 76.4939, Stdev = 61.0093	14.8–239.2	h
μ	Lognormal	Mean = 0.0362, Stdev = 0.0203	0.0113–0.0881 (GT = 7.9–61.3 h)	h^{-1}

3.3. Maximum population density (y_{max})

The maximum population density for microbial monocultures is generally regarded to be approximately 10 $\log_{10}$ colony forming units per gram (cfu/g). Because y_{max} is expressed on a logarithmic scale, the unbounded normal distribution captures the full range of potential values ($-\infty < y_{max} < +\infty$).

3.4. Initial population density (y_0)

The initial inoculum varies among experimental trials. The *L. monocytogenes* inoculum levels used by Buchanan et al. (1989) and Pin et al. (2001) ranged from 2–4 $\log_{10}$ cfu/g.

Based on the above information, priors for the growth model parameters for *L. monocytogenes* growth at 5 °C, pH 7 in broth monoculture were specified (Table 1). The prior parameter values can be combined under the Baranyi growth model (1) to obtain predictions of mean growth at time t. In this stage of the analysis, we fix $t = 240$ h (10 d) for simplicity. A sample of size $m = 30{,}000$ values for y_0, y_{max}, t_{lag}, and μ was generated using Palisades© @Risk™ (Ver. 4.5), an add-on to Microsoft© Excel™ (Ver. 9.0), and the Baranyi model was run to obtain the corresponding predicted values of mean growth after 240 h. Here, growth rather than absolute level is the desired output because it is less dependent on the initial inoculum (y_0), which varies among trials. We assume that the prior inputs are independent and allow the model and the data to induce covariance in the posterior distribution of the model parameters. Thus, the model parameters provide an implied prior on the desired output, mean growth of *L. monocytogenes* in monoculture at 5°C, pH 7 for 240 h ($\dot{g}_{240}$). The sample distribution of the implied prior is positively skewed and bimodal (with a second mode near zero due to combinations of long lag time and low growth rate values), with mean = 2.4987 $\log_{10}$ cfu/g (95% confidence interval = 0.2870 − 5.8196) (Fig. 1).

4. Stated prior on pathogen growth in broth monoculture

Buchanan et al. (1989) conducted 21 trials on *L. monocytogenes* (Scott A strain) growth in broth (TPB) monoculture at 5 °C, pH 7 under a range of conditions (aerobic and anaerobic, various salt and water activity levels). Here, we consider the results from nine trials conducted at 5 °C, pH 7 under aerobic conditions without growth inhibitors

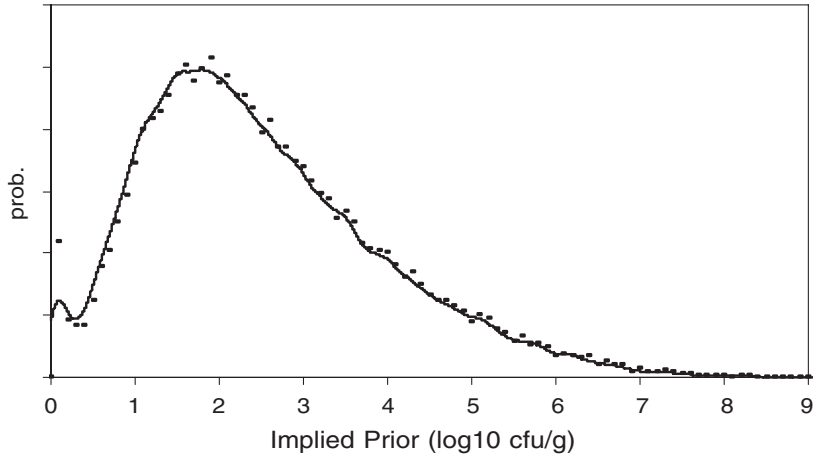

Fig. 1. Implied prior distribution for *L. monocytogenes* growth at 5 °C, pH 7, 240 h. Hash marks represent histogram bins of width 0.1 $\log_{10}$ cfu/g. Smooth curve represents non-parametric density estimate obtained by a normal (Gaussian) kernel with stdev (bandwidth) 0.1 $\log_{10}$ cfu/g.

(ComBase records: LM114_1, LM115_1, LM115_2, LM116_1, LM116_2, LM117_1, LM117_2, LM118_1, LM118_2) (Fig. 2). Linear interpolation between the two measurements bracketing $t=240$ h for each trial (indicated by the vertical reference line in Fig. 2) provides nine estimates of *L. monocytogenes* growth after 240 h (g_{240}) (Table 2). Linear interpolation (rather than model fitting) is used to maintain independence between the stated prior and information provided by the Baranyi growth model. Based on the nine estimates of g_{240}, the stated prior for *L. monocytogenes* growth in broth monoculture at 5 °C, pH 7 for 240 h is specified as: $\overline{g}_{240}$~normal (mean=4.6432, standard error (stderr)=0.3898) (note that the stated prior characterizes uncertainty about the mean response rather than natural variability about the mean).

The Buchanan et al. (1989) data also provide an estimate of variability in *L. monocytogenes* growth under replicated environmental conditions with stdev=1.1693 after 240 h of growth (Table 2). Note, however, that this variability is not constant over the growth curve (Fig. 2). Therefore, we assume that the coefficient of variation $\left(\text{cv}=\frac{\text{stdev}}{\text{mean}}\right)$ in growth is constant during the exponential growth phase (cv=0.25). This information is used below for likelihood computations and for estimating the mixed culture growth variability distribution.

5. Observed data on pathogen growth in broth monoculture under elevated CO_2

Pin et al. (2001) conducted 16 trials on *L. monocytogenes* (4b Serotype, Scott A strain) growth in broth (TSB) monoculture at 5 °C, pH 7 under a range of atmospheric conditions. Here, we consider the results from 13 trials conducted under elevated CO_2 atmospheric conditions (28–75% CO_2) (ComBase records: LB_2, LB_3, LB_4, LB_5, LB_7, LB_8, LB_9, LB_11, LB_12, LB_13, LB_14, LB_15, LB_16) (Fig. 3). Linear interpolation between the two measurements bracketing $t=240$ h (indicated by the vertical reference line in Fig. 3) provides 13 observations on *L. monocytogenes* growth after 240 h ($g_{240\text{obs}}$) with a mean of 3.0405 $\log_{10}$ cfu/g (Table 2). For the trials reported by Pin et al. (2001), variation in the observed data reflects growth under different modified atmospheric

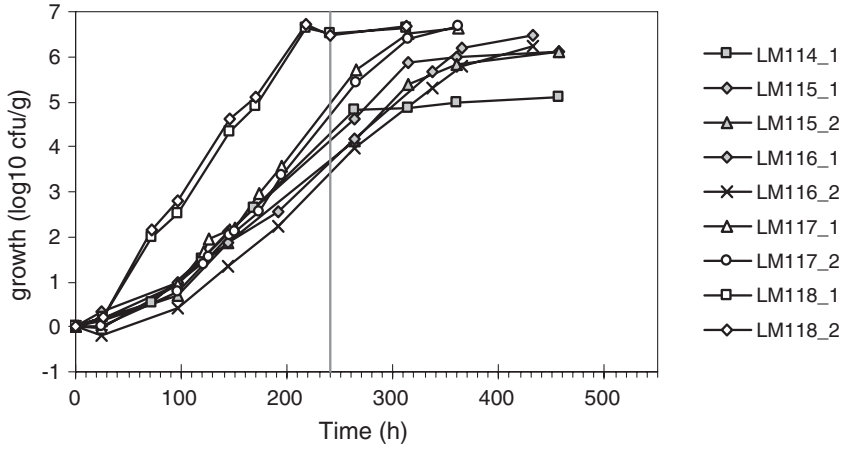

Fig. 2. Estimating stated prior on *L. monocytogenes* growth at 5 °C, pH 7, 240 h in broth monoculture. Legend key refers to ComBase Record ID for nine trials conducted without growth inhibitors under aerobic conditions (Buchanan et al., 1989).

Table 2
L. monocytogenes growth after 240 h in broth monoculture at 5 °C, pH 7

(Buchanan et al., 1989) — aerobic conditions without growth inhibitors		(Pin et al., 2001) — elevated CO_2	
ComBase record	g_{240} ($\log_{10}$cfu/g)	ComBase record	g_{240} ($\log_{10}$cfu/g)
LM114_1	4.2850	LB_2	4.2468
LM115_1	4.1220	LB_3	3.2695
LM115_2	3.6920	LB_4	3.3700
LM116_1	3.6367	LB_5	2.6074
LM116_2	3.3800	LB_7	3.0584
LM117_1	4.9665	LB_8	2.6123
LM117_2	4.7017	LB_9	2.2030
LM118_1	6.5344	LB_11	2.7707
LM118_2	6.4707	LB_12	3.1321
Mean	4.6432	LB_13	3.0222
Stdev	1.1693	LB_14	3.5570
Stderr	0.3898	LB_15	3.1788
cv	0.2518	LB_16	2.4985
		Mean	3.0405

conditions rather than intrinsic variability in pathogen growth under replicated conditions.

6. Bayesian synthesis

This paper employs a variant of the Bayesian synthesis method based on Green et al. (1999). The Bayesian synthesis method is used to update the joint distribution of two Baranyi model parameters–lag time and maximum specific growth rate—for growth of *L. monocytogenes* in monoculture at 5 °C, pH 7 under elevated carbon dioxide (CO_2) levels. The information presented above is synthesized using the following algorithm.

a. Compute the density of the pathogen growth model predictions ($\hat{g}_{240}$) on the implied prior.

Because the implied prior distribution is skewed and bimodal (Fig. 1), the density of the model predictions was estimated using a kernel estimator, a widely used non-parametric data smoothing technique (Bowman and Azzalini, 1997). The density of each of the $m=30{,}000$ sample values of $\hat{g}_{240}$ on the implied prior was estimated using a normal (Gaussian) kernel with a bandwidth (stdev) of 0.1 $\log_{10}$ cfu/g. The procedure was performed using the *sm.density* function from the *sm library*, a suite of Insightful© S-PLUS™ routines (Bowman et al., 2000).

b. Compute the density of the pathogen growth model predictions ($\hat{g}_{240}$) under the stated prior ($\overline{g}_{240}$).

The density of each of the $m=30{,}000$ sample values of $\hat{g}_{240}$ under the stated prior ($\overline{g}_{240}$~normal (mean=4.6432, stderr=0.3898)) was obtained using the Microsoft© Excel Normdist function and normalizing the densities to sum to 1.

c. Compute the likelihood of the pathogen growth model predictions given the observed data on *L. monocytogenes* growth under elevated CO_2 ($L(\hat{g}_{240}|g_{240\text{obs}})$).

The empirical data used to update the monoculture growth model are the 13 observations under elevated CO_2 reported by Pin et al. (2001). For the likelihood computations, we estimate the standard deviation is equal to 0.7601, based on a mean of 3.0405 and assuming cv=0.25. (The cv value is based on the Buchanan et al. (1989) replicate trials.) In general, the likelihood of the model prediction given the observed data is estimated as:

$$L(\hat{g}_{240}|g_{240\text{obs}}) = \prod_{n=1}^{16} \text{Normal}(g_n|\text{mean} = \hat{g}_{240}, \text{stdev}=0.7601) \quad (2)$$

The likelihoods for the $m=30{,}000$ simulated sample values are obtained and normalized.

Simulated parameter combinations (y_0, $y_{\max}$, t_{lag}, μ) derived from the priors that imply growth levels not supported by the growth data from elevated CO_2 trials reported by Pin et al. (2001) result in very small likelihoods (here, normalized likelihood values ranged from 4.41E−196–3.05E−04).

d. Compute importance sampling weights.

Let $q_\theta(g)$ represent the prior distribution on the output implied by the pathogen growth model parameter estimates (obtained in step 6a, with $\theta=y_0, y_{\max}, t_{\text{lag}}, \mu$). Let $q_g(g)$ represent

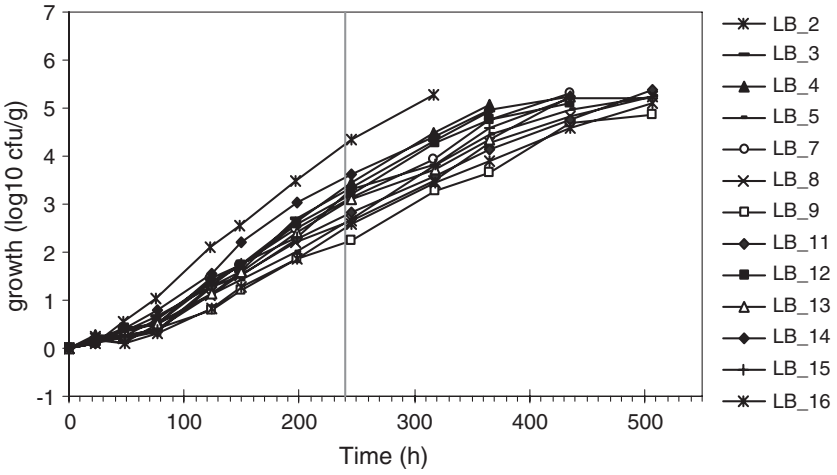

Fig. 3. Observed data on *L. monocytogenes* growth at 5 °C, pH 7, 240 h in broth monoculture under modified atmospheric conditions. Legend key refers to ComBase Record ID for 13 trials conducted with elevated carbon dioxide levels (Pin et al., 2001).

the independent stated prior distribution on the output (obtained in step 6b). Geometric pooling of $q_\theta(g)$ and $q_g(g)$ yields:

$$q_\theta^p(g) \propto q_\theta(g)^\alpha q_g(g)^{1-\alpha} \quad (3)$$

where $q_\theta^p(g)$ is the pooled distribution. We must have $\alpha=0.5$ to ensure that the method is invariant to relabeling of inputs and outputs. That is, we obtain the same result running the model "forwards" or "backwards" (note that the stated prior on the outputs could be used to develop an implied prior on the inputs by running the model backwards. But the implied input distributions will not be identical to specified priors for the input parameters. Pooling the implied and stated prior distributions avoids the incoherence of two different "priors" for both the inputs and outputs). In addition, setting $\alpha=0.5$ places equal weight on both the implied and stated priors (Green et al., 1999).

A posterior distribution for the output ($\pi(g)$) could be developed using $q_\theta^p(g)$. The posterior distribution for the Baranyi growth model parameters ($\pi(\theta)$) is not directly available from this posterior, however, because numerous parameter value combinations ($Z=\theta_1, \theta_2, \ldots$) may obtain the same output value (g_k). This problem is solved by assigning a collective probability for all parameter combinations that yield a given output value equal to the pooled density ($q_\theta^p(g_k)$) and allocating the collective probability among parameter value combinations in proportion to the prior distribution on θ ($q_\theta(\theta_Z)$) (Green et al., 1999):

$$q_\theta^p(\theta_Z) = q_g^p(g_k)\frac{q_\theta(\theta_Z)}{q_\theta(\theta_k)} \quad (4)$$

According to (3), with $\alpha=0.5$

$$q_g^p(g_k) \propto q_\theta(g_k)^{0.5} q_g(g_k)^{0.5} \quad (5)$$

and therefore

$$q_\theta^p(\theta_Z) \propto q_\theta(\theta_Z)\left(\frac{q_g(g_k)}{q_\theta(g_k)}\right)^{0.5} \quad (6)$$

Thus the posterior distribution for Baranyi growth model parameters can be obtained:

$$\pi(\theta) \propto q_\theta^p(\theta) L(\hat{g}|g_{\text{obs}}) \quad (7)$$

Importance sampling weights are computed as follows (Green et al., 1999):

$$w_m = \left(\frac{q_g(g)}{q_\theta(g)}\right)^{0.5} L(\hat{g}_{240}|g_{240\text{obs}}) \quad (8)$$

Note that if the implied and stated densities are identical, then the importance weights are equal to the likelihood of the model predictions given the new data (here, normalized weight values ranged from 2.44E−208 – 3.66E−04).

e. Sample values from the joint input and implied output distribution with probabilities proportional to w_m.

Sampling/Importance Resampling (SIR) is a noniterative algorithm used to simulate Bayesian posterior distributions (Rubin, 1988). (This procedure is also referred to as a weighted bootstrap (Smith and Gelfand, 1992).) In SIR, m samples are drawn from an initial approximation to the desired distribution, and then $l<m$ samples are randomly drawn from the first finite sample (m) with probability proportional to their importance (i.e., sampling weight). The rationale of the SIR algorithm is based on the fact that as $m/l\rightarrow\infty$, the l sample values represent independent draws from the desired posterior distribution (Rubin, 1988). In practice, the choice of a ratio m/l depends on the adequacy of the initial approximation. If the initial approximation is perfect, then $m/l=1$ is proper, but as the initial approximation to the posterior gets poorer, m/l must increase. Rubin (1987) suggests that a ratio of $m/l=20$ will often be more than adequate. Therefore in this analysis (with $m=30{,}000$), $l=1500$ values from the simulated joint distribution $q(\theta,\hat{g}_{240})$ are drawn using Monte Carlo simulation from the empirical distribution with the normalized importance sampling weights obtained in the previous step. The resulting samples are approximate samples from the geometrically pooled posterior distributions for the pathogen growth model inputs and output $p(\theta,\hat{g}_{240})$.

Monte Carlo simulation was performed with Latin Hypercube sampling (1,500 iterations) using @Risk™. Latin Hypercube sampling involves stratified sampling without replacement in which the model input distributions are split into l intervals of equal probability. In comparison to simple random sampling, Latin Hypercube sampling ensures a representative sample is drawn from all parts of an input distribution (Vose, 2000). Once the Bayesian synthesis model was prepared, the Monte Carlo simulation itself required less than three minutes on a 2.40 GHz cpu personal computer. However, model set-up, including generation of the model input values and calculation of the importance sampling weights, requires substantially more time and considerable effort.

In sum, Bayesian synthesis of the informed priors on the Baranyi growth model parameters, the stated prior based on Buchanan et al. (1989), and the observed data based on Pin et al. (2001) results in updated distributions of the model inputs—y_0, $y_{\max}$, t_{lag}, and μ—and the model output—growth of *L. monocytogenes* at 5 °C, pH 7 after 240 h in broth monoculture under elevated CO_2 levels.

7. Observed data on pathogen growth in food with mixed microbial populations

The Veterinary Faculty of Universidad Complutense, Madrid provided data from a trial on *L. monocytogenes* (4b Serotype, Scott A strain) grown in competition with natural microflora in fish (hake) at 5°C, pH 6.8 under modified atmospheric conditions (43% CO_2, 57% N_2) (ComBase record P_11L, Table 3). These observed data are used to update the Baranyi model for *L. monocytogenes* growth in mixed microbial populations using the SIR algorithm.

8. Update model for pathogen growth in food with mixed microbial populations

The first step in the SIR algorithm is to "obtain a decent first pass approximation" to the desired distribution (Rubin, 1988).

Table 3
L. monocytogenes growth under competition in Hake at 5°C, pH 6.8, 43% CO_2, 57% N_2

Time (h)	$\log_{10}$ cfu/g
0.00	3.490
72.42	3.980
120.07	4.230
168.49	4.590
240.03	5.760
288.09	5.100
336.07	5.740
408.10	6.410

Source: ComBase record P_11L.

The updated joint distribution of t_{lag} and μ obtained by Bayesian synthesis of broth monoculture data provides a reasonable first approximation to these input parameters for modeling *L. monocytogenes* growth in food with mixed microbial populations under similar environmental conditions (i.e., temperature, pH, atmosphere). The updated joint distribution for t_{lag} and μ reflects the correlation in the posterior distribution of the Baranyi model parameters induced by the Bayesian synthesis procedure. In this manner, we have used the Bayesian synthesis procedure to draw inferences from broth monoculture data to obtain an informed prior for modeling pathogen growth in food with mixed microbial populations.

Buchanan and Bagi (1999) demonstrated, however, that *L. monocytogenes* grown in co-culture with a spoilage organism (*Pseudomonas fluorescens*) can attain maximum population densities that are lower, higher, or the same compared to levels of the pathogen in monoculture, depending on the temperature, acidity, and availability of water in the surrounding environment. Therefore, in order to model the growth of *L. monocytogenes* in food with mixed microbial populations, we combine the updated joint distribution of t_{lag} and μ obtained by the Bayesian synthesis procedure with prior distributions for y_0 and y_{max} appropriate for mixed culture conditions.

Priors for the Baranyi model parameters for *L. monocytogenes* growth in fish with mixed microbial populations at 5 °C, pH 6.8 under elevated CO_2 conditions were specified as in Table 4. The *L. monocytogenes* inoculum (y_0) in the U. Madrid fish trial (ComBase record P_11L) is 3.49 $\log_{10}$cfu/g (Table 3), with measurement was by colony counts. A normal distribution with mean=3.49 and stdev=0.1745 (i.e., cv=0.05, 95% confidence interval=3.15−3.83 $\log_{10}$ cfu/g) captures the full range of potential values for y_0 (by comparison, according to the U.S. Environmental Protection Agency drinking water laboratory quality assurance criteria, colony counts from the same sample by two or more analysts should agree within 10% (EPA, 1997)).

The U. Madrid fish trial (ComBase record P_11L, Table 3) contains no data to indicate the stationary phase of the growth curve. Such microbial growth experiments are not uncommon (e.g., Fig. 3), and this may arise because the study objective is to estimate the maximum specific growth rate or time until a specified amount of growth occurs. In such cases, however, an algorithm fitting a sigmoid curve to the data may fail (e.g., by generating nonsensical y_{max} parameter values). Baranyi et al. (1993) suggest that the solution can be either to fix y_{max} or to model only the lag and exponential phase of growth. Instead, we exploit prior information to characterize uncertainty about the complete growth curve. Here, the prior distribution for y_{max} for pathogen growth in mixed microbial populations was simulated as dependent on y_0, with y_{max}~uniform(min=y_0, max=11 $\log_{10}$cfu/g) to account for the full range of potential outcomes of interspecific competition (the simulated prior distribution for y_{max} has mean=7.2381, stdev=2.1686 ($\log_{10}$ cfu/g)). Finally, the joint prior distribution of t_{lag} and μ was generated using the updated marginal distributions obtained by Bayesian synthesis of the monoculture data and specifying the empirically estimated correlation from the updated joint distribution ($r(t_{lag},\mu)$=0.83). A joint prior distribution on all model inputs (y_0, y_{max}, t_{lag}, and μ) was generated by Monte Carlo simulation (sample size m=30,000) using @Risk™.

Simulated parameter combinations ($\theta=y_0$, y_{max}, t_{lag}, μ) can be combined under the Baranyi growth model (1) to predict mean growth at any time ($\bar{g}_t$) and thus obtain an implied growth curve over time. Because the U. Madrid fish trial (ComBase record P_11L) does not contain replicates, we estimate the standard deviation assuming cv=0.25 (calculated based on observed growth, g_{tobs}) for the likelihood computations. In general, the likelihood given the observed data is estimated as:

$$L(\theta|g_{obs}) = \prod_{t_0}^{t_7} \text{Normal}(g_{tobs}|mean = \bar{g}_t, \text{stdev} = 0.25*g_{tobs}) \quad (9)$$

The normalized likelihood values provide importance sampling weights for the m=30,000 parameter combinations. Simulated parameter combinations (θ) that imply growth curves not supported by the U. Madrid fish data result in very small likelihoods (here, normalized likelihood values ranged from 1.58E−196–1.83E−02). A joint posterior distribution on the

Table 4
Prior distributions for Baranyi growth model input parameters for *L. monocytogenes* grown in competition with natural microflora in fish (Hake) at 5 °C, pH 6.8, 43% CO_2, 57% N_2

Input	Distribution	Dist. parameters	95% confidence interval	Units
y_0	Normal	Mean=3.49, Stdev=0.349	2.8060–4.1740	$\log_{10}$ cfu/g
y_{max}	Uniform	Min=y_0, Max=11	3.5897–10.8128	$\log_{10}$ cfu/g
t_{lag}	Lognormal	Mean=53.8320, Stdev=29.4714	17.3–128.8	h
μ	Lognormal	Mean=0.0419, Stdev=0.0098	0.0227–0.0611	h^{-1}
		$r(t_{lag},\mu)$=0.83	(GT=11.3–30.6 h)	

Table 5
Prior and posterior distributions for lag time (t_{lag}) and maximum specific growth rate (μ) in Bayesian synthesis of broth monoculture data

	t_{lag} (h)		μ (h^{-1})	
	Prior	Posterior	Prior	Posterior
Mean	76.4939	53.8320	0.0362	0.0419
Stdev	61.0093	29.4714	0.0203	0.0098
cv	0.7976	0.5475	0.5608	0.2339
% change mean	−30%		+16%	
% change cv	−31%		−58%	

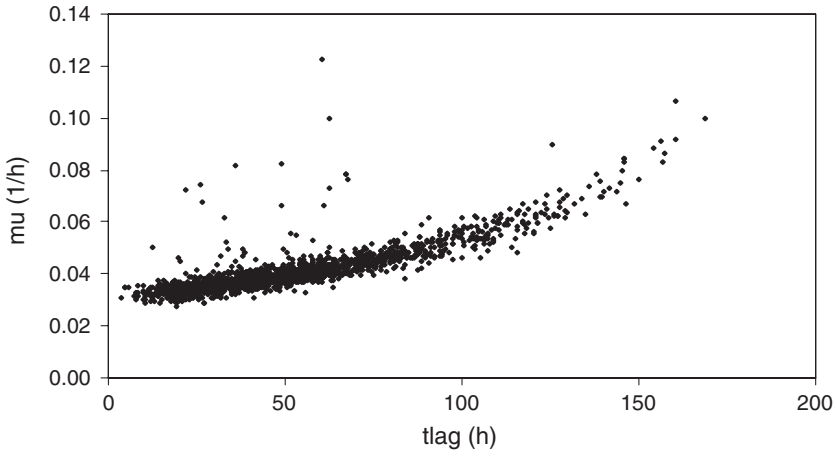

Fig. 4. Updated joint distribution of lag time (t_{lag}) and maximum specific growth rate (μ) obtained by Bayesian synthesis of *L. monocytogenes* broth monoculture data.

Baranyi growth model inputs and outputs is then generated by importance sampling, with $l=1{,}500$ values drawn using Monte Carlo simulation from the empirical distribution. Monte Carlo simulation was performed with Latin Hypercube sampling (1500 iterations) using @Risk™. The updated inputs represent the posterior joint uncertainty distribution for the Baranyi growth model parameters ($\pi(\theta)$). The updated outputs represent the posterior uncertainty distribution of mean growth at time t ($\pi(g_t)$), i.e., uncertainty about the mean growth curve.

Uncertainty about a given percentile of the growth variability distribution at time t ($g_{t\%ile}$) was estimated by the inverse normal distribution, assuming cv = 0.25 (based on the Buchanan et al. (1989) data):

$$g_{t\%ile} \sim \text{NormInv}(\%ile|\text{mean} \sim \pi(g_t), \text{stdev} \sim 0.25*\pi(g_t)) \quad (10)$$

The $g_{t\%ile}$ distribution was generated by fixing the percentile value of the normal variability distribution (e.g., at 95th %ile) and drawing random samples with replacement from the empirical posterior output uncertainty distribution ($\pi(g_t)$). This bootstrap (Efron and Tibshirani, 1993) procedure was performed by Monte Carlo simulation with Latin Hypercube sampling (10,000 iterations) using @Risk™.

9. Sensitivity analysis

To analyze the sensitivity of the posterior distributions for *L. monocytogenes* growth in fish with mixed microbial populations to the specified prior on the model inputs, we ignore the results of the broth monoculture data analysis and repeat the SIR algorithm using the pre-Bayesian synthesis prior distributions for t_{lag} and μ obtained in Sec. 3 (Table 1). The joint prior distribution on y_0 and y_{max} was fixed at the same values obtained in Sec. 8 (Table 4).

10. Results

10.1. Bayesian synthesis of L. monocytogenes broth monoculture data

The results of the Bayesian synthesis can be regarded as posterior distributions constrained to agree with the specified Baranyi growth model. The procedure resulted in posterior input distributions for $\pi(y_0)$ and $\pi(y_{max})$ that were unchanged from their priors. In contrast, the posterior input distributions $\pi(t_{lag})$ and $\pi(\mu)$ were revised substantially (Table 5). The lag time parameter underwent the larger relative change in central location, while the reduction in relative spread was greater for

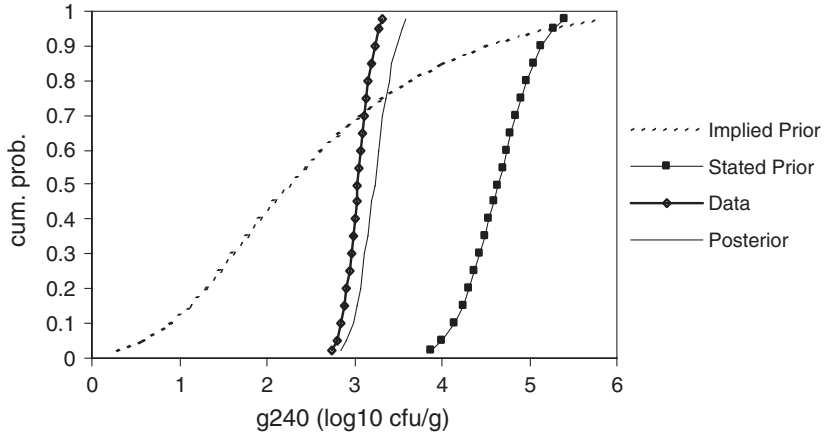

Fig. 5. Bayesian synthesis: uncertainty distributions for mean *L. monocytogenes* growth at 5 °C, pH 7, 240 h in broth monoculture.

the growth rate parameter. With the exception of the posterior joint distribution $\pi(t_{lag}, \mu)$ (Fig. 4, with $r = 0.83$), the correlation induced by the Bayesian synthesis procedure between the Baranyi growth model input parameters was negligible ($|r \leq 1|$). The updated 95% confidence interval for GT (Table 4) contains the point estimate of 29 h for *L. monocytogenes* at 5 °C, pH 7 provided by the food microbiology experts (Food and Agriculture Organization of the United Nations, 1999).

Fig. 5 presents uncertainty distributions of mean growth of *L. monocytogenes* in broth monoculture at 5 °C, pH 7 after 240 h

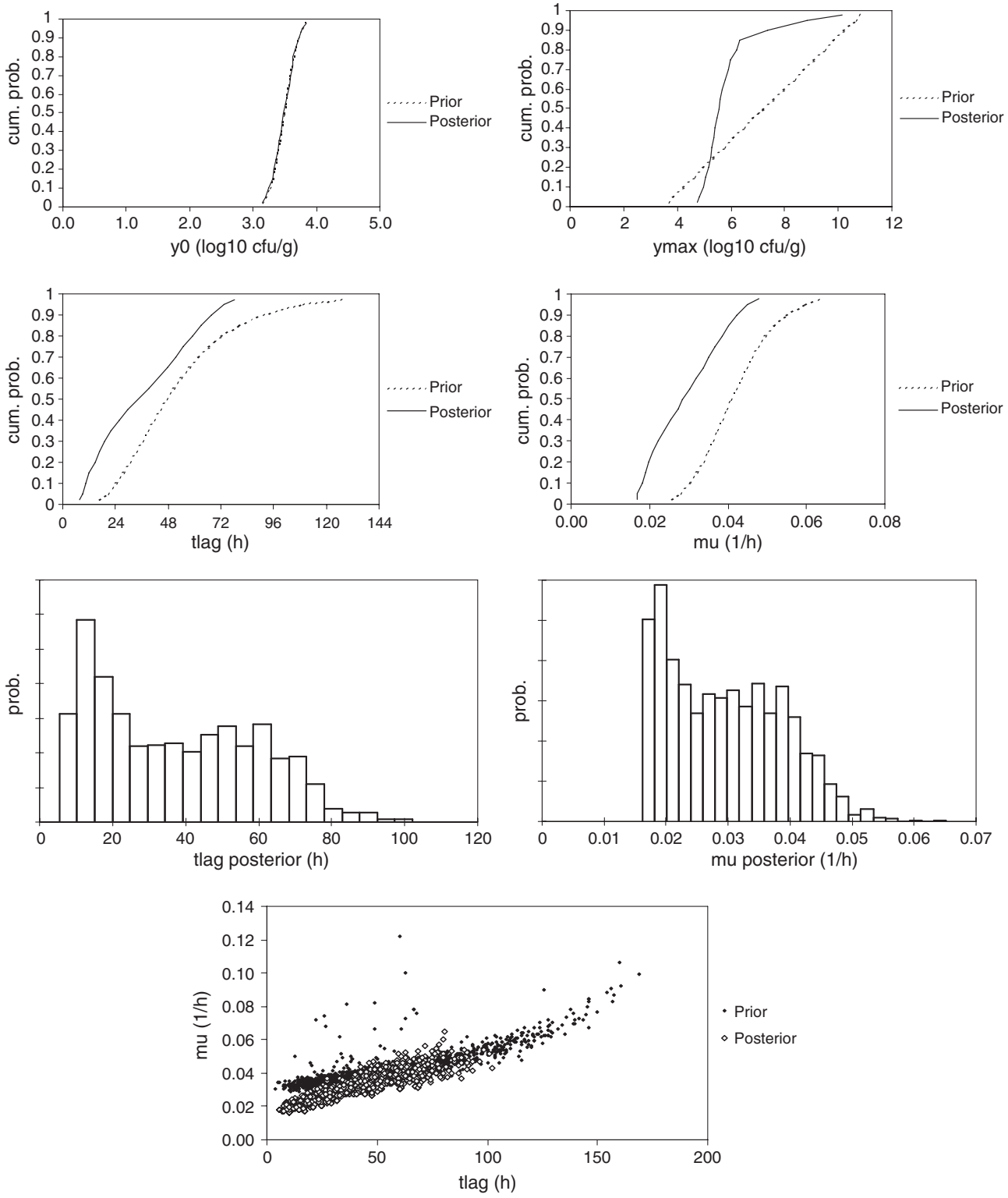

Fig. 6. Prior and posterior distributions of Baranyi model input parameters for *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2.

Table 6
Summary of posterior distribution of Baranyi model input parameters for *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2

Input (units)	Mean	Stdev	95% Confidence interval	Correlation matrix (r)			
				y_0	y_{max}	t_{lag}	μ
y_0 ($\log_{10}$cfu/g)	3.5	0.2	3.1–3.8	1			
y_{max} ($\log_{10}$cfu/g)	5.9	1.2	4.7–10.1	0.22	1		
t_{lag} (h)	37.1	22.0	7.8–78.8	−0.05	−0.47	1	
μ (h^{-1})	0.0295	0.0093	0.0168–0.0479 (GT=14.5–41.2 h)	−0.02	−0.53	0.92	1

for the implied prior, stated prior, the observed data used to update the model, and the posterior resulting from the Bayesian synthesis procedure. The posterior output distribution $\pi(g_{240})$ (mean=3.2, stderr=0.2 ($\log_{10}$ cfu/g)) falls closer to the data used to update the model (from Pin et al., 2001) than to stated prior (from Buchanan et al., 1989). It can be shown that if the situation were reversed—the stated prior was obtained from Pin et al. (2001), and the data from Buchanan et al. (1989) were used to update the model—the posterior would fall closer to the results of Buchanan et al. (1989). In this application, however, the posterior is intended to reflect *L. monocytogenes* growth at elevated CO_2 levels (as in Pin et al., 2001) rather than aerobic conditions (as in Buchanan et al., 1989). The Bayesian synthesis procedure, however, permits characterization of pathogen growth at elevated CO_2 levels while incorporating information available from Buchanan et al. (1989) (e.g., uncertainty and variability in growth under replicated environmental conditions) not provided by Pin et al. (2001). The Bayesian synthesis procedure also results in information about the correlation (or the lack thereof) between Baranyi growth model parameters that is used to generate the initial approximation of *L. monocytogenes* growth required by the SIR algorithm used to model the pathogen behavior in mixed culture.

10.2. L. monocytogenes in fish with mixed microbial populations and elevated CO_2

Fig. 6 compares the prior and posterior distributions for the Baranyi growth model parameters (including the joint distribution of t_{lag} and μ), and Table 6 summarizes the updated input parameters for *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2. The posterior input distribution $\pi(y_{max})$ indicates very large uncertainty in maximum pathogen density (the 95% confidence interval for y_{max} spans >5 orders of magnitude), consistent with simulation of a simple theoretic model of interspecific microbial competition (Powell et al., 2004). The posterior input distributions $\pi(t_{lag})$ and $\pi(\mu)$ both were revised downward and tightened. A strong correlation (r=0.92) remains in the posterior joint distribution of lag time and maximum specific growth rate ($\pi(t_{lag}, \mu)$). (Note that the prior joint distribution for t_{lag} and μ in Fig. 6 is the posterior joint distribution obtained by Bayesian synthesis of *L. monocytogenes* broth monoculture data shown in Fig. 4.) Indeed, the correlation between the lag time and maximum specific growth rate parameters of the Baranyi growth model (1) appears to be inherent to the model (Grijspeerdt and Vanrolleghem, 1999). The updated 95% confidence interval for GT (Table 6) contains the point estimate of 29 h for *L. monocytogenes* at 5°C, pH 7 provided by the food microbiology experts (Food and Agriculture Organization of the United Nations, 1999).

Fig. 7 summarizes the posterior output distribution $\pi(g_t)$, presenting 5% and 95% confidence levels in mean growth of *L. monocytogenes* in competition with natural microflora in fish (hake) at 5 °C, pH 6.8, 43% CO_2, 57% N_2. (The 5% and 95% confidence levels are the lower and upper bounds, respectively, of a 90% confidence interval.) At 230 h (9.6 d), the 95% confidence level for the 95th percentile of the

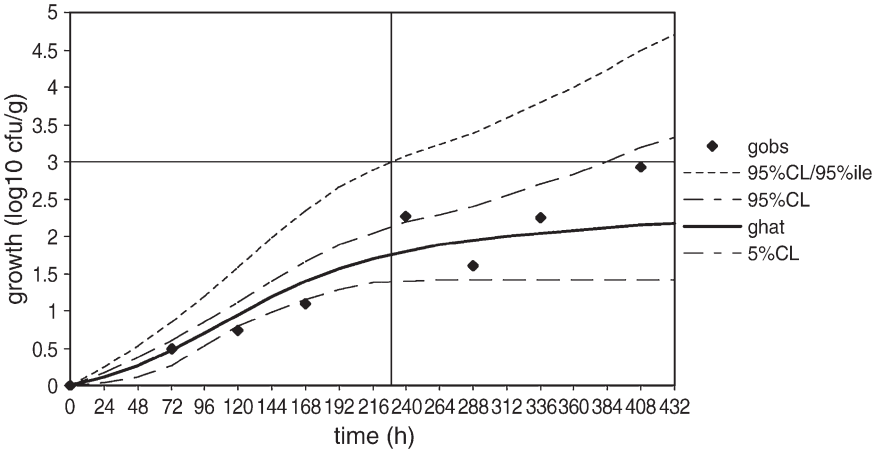

Fig. 7. Estimated uncertainty and variability in *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2.

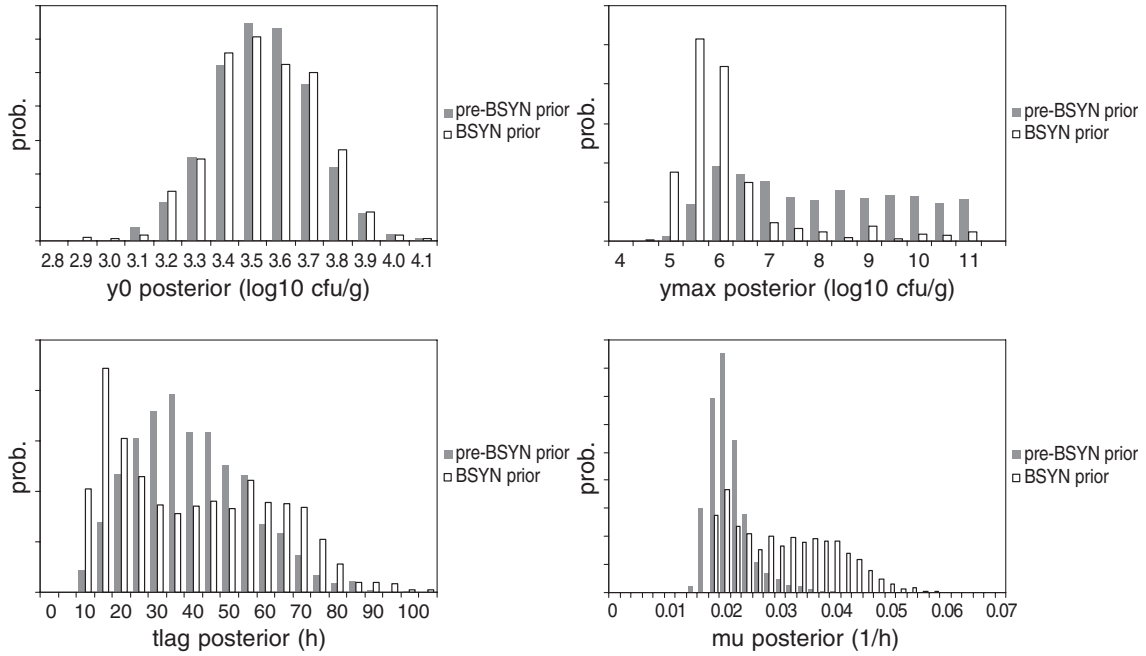

Fig. 8. Alternative posterior distributions of Baranyi model input parameters for *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2. Legend key refers to alternative prior distributions for t_{lag} and μ discussed in Sec. 9.

estimated growth variability distribution exceeds 3 $\log_{10}$ cfu/g (indicated by the horizontal and vertical reference lines in Fig. 7).

10.3. Sensitivity analysis

Fig. 8 compares the posterior distributions for the Baranyi growth model parameters obtained using the pre-Bayesian synthesis prior distributions for t_{lag} and μ (pre-BSYN prior) with those resulting from specifying the prior using the updated joint distribution of t_{lag} and μ obtained by Bayesian synthesis of broth monoculture data (BSYN prior). In particular, the comparison suggests that the Bayesian synthesis of the broth monoculture data may provide an overconfident estimate of μ, resulting in an insufficiently diffuse prior for updating the model based on the U. Madrid fish trial (ComBase record P_11L). Alternatively, the broth monoculture data analysis may be interpreted as providing a better estimate of the maximum specific growth rate intrinsic to *L. monocytogenes* due to the greater quantity of data and the absence of interspecific competition. Table 7 summarizes the alternative posterior distributions (obtained with the pre-BSYN prior) for the Baranyi model parameters for *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2. Note that the updated 95% confidence interval for GT contains the point estimate of 29 h for *L. monocytogenes* at 5°C, pH 7 provided by the food microbiology experts (Food and Agriculture Organization of the United Nations, 1999).

Although the posterior marginal uncertainty distributions for the growth model parameters overlap (comparing Tables 6 and 7), the results indicate that the joint posterior distribution of the model inputs is sensitive to the choice of priors for t_{lag} and μ. This can be seen in the different shape of the growth curves in Figs. 7 and 9. The latter summarizes the posterior output distribution $\pi(g_t)$ under the alternative prior. Nevertheless, a decision based on the time until 3 $\log_{10}$ cfu/g growth would not be very sensitive to the uncertainty about the growth curve form associated with specification of the prior. In comparison to the previous estimate of 230 h (9.6 d) (Fig. 7), the 95% confidence level for the 95th percentile of the alternative growth variability distribution exceeds 3 $\log_{10}$ cfu/g at 263 h (11d) (indicated by the horizontal and vertical reference lines in Fig. 9). Similarly, a decision based on the

Table 7
Summary of posterior distribution of Baranyi model input parameters for *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2 under alternative prior

Input (units)	Mean	Stdev	95% Confidence interval	Correlation matrix (r)			
				y_0	y_{max}	t_{lag}	μ
y_0 ($\log_{10}$cfu/g)	3.5	0.2	3.1–3.8	1			
y_{max} ($\log_{10}$cfu/g)	7.8	1.7	5.3–10.8	0.02	1		
t_{lag} (h)	37.2	15.7	11.7–71.0	−0.02	−0.11	1	
μ (h^{-1})	0.0197	0.0038	0.0146–0.0301 (GT=23.0–47.4 h)	0.03	−0.39	0.58	1

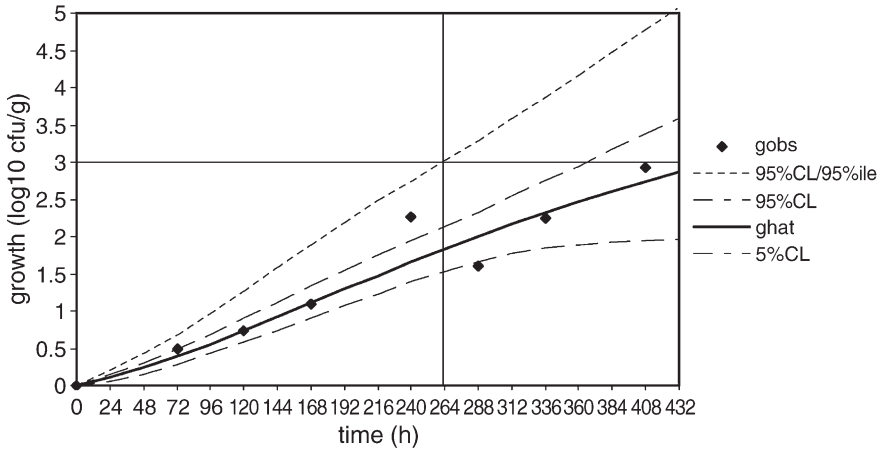

Fig. 9. Estimated uncertainty and variability in *L. monocytogenes* growth in competition with natural microflora in fish at 5 °C, pH 6.8, 43% CO_2, 57% N_2 under alternative prior.

time until 1 $\log_{10}$ cfu/g growth would differ by just 16 h (83 h under the BSYN prior (Fig. 7) vs. 99 h under the pre-BSYN prior (Fig. 9).

11. Discussion

The SIR algorithm comes with "one important caveat" (Smith and Gelfand, 1992). The choice of a ratio m/l depends on the goodness of the first pass approximation to the posterior distribution (Rubin, 1988). But, it is unclear how we know if the first pass approximation is decent or whether we always need $m \gg l$ (Dunsmore, 1988). SIR computational intensity increases the less the prior resembles the posterior, because a larger initial sample size m will need to be drawn to adequately estimate the posterior. In this paper, we have used Bayesian synthesis (Raftery et al., 1995) of assorted relevant information to be confident in obtaining a "decent first pass approximation" to the growth of *L. monocytogenes* under competitive growth conditions. In general, any particular data set provides partial information about the system being investigated under the specific conditions under which the data were generated. In cases where a "gold standard" is unavailable, performance measures based on model validation concepts provide little guidance about how to draw reasonable inferences from available, imperfect information. In cases where ideal data are available, they may be of limited quantity, provide little information about the underlying biological process, or both. In either case, Bayesian statistical methods such as Bayesian synthesis (Raftery et al., 1995) and SIR (Rubin, 1988) provide useful analytical tools for integrating available information from disparate sources to develop models for use in microbial pathogen risk assessment.

Given the paucity of experimental data available on pathogen growth under competitive conditions, it may be tempting to generalize from this analysis to make broader inferences about *L. monocytogenes* growth in foods with mixed microbial populations. It should be noted, however, that the U. Madrid fish trial (ComBase record P_11L) occurred under specific abiotic and *biotic* conditions. The path and outcome of complex microbial community dynamics may vary dependent on microbial composition, initial densities, and the sequence of contamination events (Powell et al., 2004). Analytical methods for synthesizing the typically motley assortment of data available for microbial risk assessment provide a measure of protection but are no panacea for overconfidence.

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